	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	2	hmgi SAME dna	USPAT; Derwent	2000/11/03 14:29			0

DERWENT-ACC-NO: 1997-333837
DERWENT-WEEK: 200031

TITLE: DNA sequences representing aberrant forms of human high mobility group
protein genes - useful for treatment of endometriosis and tumours, or for
modulating vascularisation, etc

INVENTOR: BULLERDIEK, J

PATENT-ASSIGNEE: BULLERDIEK J[BULLI]

PRIORITY-DATA: 1995DE-1048122 (December 21, 1995)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2000504933	April 25, 2000	N/A	120	C12N 015/09
W	June 26, 1997	N/A	058	C12N 015/63
DE 19548122 A1	July 3, 1997	G	160	C12N 015/12
WO 9723611 A2	July 17, 1997	N/A	000	C12N 015/12
AU 9718705 A	October 2, 1997	N/A	000	C12N 015/63
WO 9723611 A3	October 14, 1998	G	000	C12N 015/12
EP 870024 A2				

DESIGNATED-STATES: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DK EE ES FI GB G
E HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT
RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN AT BE CH DE DK EA ES FI FR GB GR
IE IT KE LS LU MC MW NL OA PT SD SE SZ UG AT BE CH DE DK ES FI FR GB GR IE IT L
I LU NL PT SE

CITED-DOCUMENTS: 4.Jnl.Ref; EP 727487

APPLICATION-DATA:

PUB-NO	APPL-DESCRIPTOR	APPL-NO	APPL-DATE

JP2000504933W December 20, 1996	N/A	1996WO-DE02494
JP2000504933W December 20, 1996	N/A	1997JP-0523225
JP2000504933W N/A	Based on	WO 9723611
DE 19548122A1 December 21, 1995	N/A	1995DE-1048122
WO 9723611A2 December 20, 1996	N/A	1996WO-DE02494
AU 9718705A December 20, 1996	N/A	1997AU-0018705
AU 9718705A N/A	Based on	WO 9723611
WO 9723611A3 December 20, 1996	N/A	1996WO-DE02494
EP 870024A2 December 20, 1996	N/A	1996EP-0946114
EP 870024A2 December 20, 1996	N/A	1996WO-DE02494
EP 870024A2 N/A	Based on	WO 9723611

INT-CL (IPC): A61K031/70; A61K031/713 ; A61K038/00 ; A61K038/17 ;
A61K039/395 ; A61K048/00 ; A61P009/10 ; A61P015/02 ; A61P015/18 ;
A61P035/00 ; C07K014/47 ; C07K014/82 ; C07K016/18 ; C07K019/00 ;
C12N001/19 ; C12N001/21 ; C12N005/10 ; C12N015/02 ; C12N015/09 ;
C12N015/11 ; C12N015/12 ; C12N015/63 ; C12N015/85 ; C12N015/87 ;
C12P021/08 ; C12Q001/68 ; C12N015/09 ; C12R001:91

ABSTRACTED-PUB-NO: DE 19548122A
BASIC-ABSTRACT: Novel DNA sequences (A), of between 349 and 850 bp (19
sequences in total), represent at least parts of aberrant forms of the human
high mobility group protein (HMG) genes, designated HMGI-C and located on
chromosome 12. All sequences are given in the specification. Also new are:
(1) expression vectors containing (A), under promoter control; (2) host cells
containing such vectors; (3) proteins (I), including mutants, fragments and
(non-)chemically modified forms, encoded by (A); and (4) use of a MAG (multiple
tumour aberration growth) gene or HMG gene for modulation of

vascular
development.

USE - (Anti)sense, single and/or double stranded DNA or RNA (A), poly- or
mono-clonal antibodies (Ab), their fragments or derivatives against (I),
translation products of (A) or expression modulators of (A) are used in kits
(claimed) for modulating vascular development. The kits are particularly used
in human or veterinary medicine, to reduce, block or stimulate angiogenesis
(particularly in tumours) or vascularisation (particularly to treat or prevent
blindness caused by neo-vascularisation), also to improve vascular provision in
myocardium damaged by infarction. Also the kits can be used for treatment of
endometriosis and tumours, for contraception (local or oral) and for tissue
regeneration, especially in degenerating or damaged tissue, particularly
mesenchymal tissue (cartilage, muscle, adipose, connective or supporting
tissues), both in vivo and in vitro (added to cell, tissue or organ cultures).
Typical applications of regeneration are in treatment of arthritis and muscular
dystrophy, but the method can also be used cosmetically.

ADVANTAGE - Because of their specific actions, (A) etc. avoid the side effects
associated with standard methods for treating the specified diseases. The
regeneration method can be applied to tissues which are currently impossible or
difficult to regenerate, and use of biological materials (with attendant risks
of viral transmission and anaphylactic shock) is avoided.

CHOSEN-DRAWING: Dwg.0/19

TITLE-TERMS:
DNA SEQUENCE REPRESENT FORM HUMAN HIGH MOBILE GROUP PROTEIN GENE USEFUL TREAT
TUMOUR MODULATE

DERWENT-CLASS: B04 C06 D16

CPI-CODES: B04-E02F; C04-E02F; B04-E03F; C04-E03F; B04-E05;

C04-E05; B04-E06;
C04-E06; B04-E08; C04-E08; B04-F0100E; C04-F0100E; B04-G01;
C04-G01; B04-G21;
C04-G21; B04-G22; C04-G22; B04-N02; C04-N02; B04-N0200E;
C04-N0200E; B11-C07A;
C11-C07A; B11-C08E; C11-C08E; B12-K04A; C12-K04A; B14-C09;
C14-C09; B14-F02;
C14-F02; B14-H01; C14-H01; B14-L01; C14-L01; B14-N14; C14-N14;
B14-P01B;
C14-P01B; D05-H09; D05-H12A; D05-H12E; D05-H17A6;

CHEMICAL-CODES:
Chemical Indexing M1 *01*
    Fragmentation Code
    M423 M424 M710 M740 M903 N103 P420 P421 P633 P831
    P842 Q233 V753

Chemical Indexing M1 *02*
    Fragmentation Code
    M423 M710 M903 N135 Q233 V752 V754

Chemical Indexing M6 *03*
    Fragmentation Code
    M903 P420 P421 P633 P831 Q233 R515 R521 R627

SECONDARY-ACC-NO:
CPI Secondary Accession Numbers: C1997-107320

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=> d his all

     (FILE 'HOME' ENTERED AT 14:22:08 ON 03 NOV 2000)

     FILE 'BIOSIS, EMBASE, MEDLINE, LIFESCI, SCISEARCH, CAPLUS' ENTERED AT
     14:22:23 ON 03 NOV 2000
L1           326 S HMGI(P)DNA
L2             3 S L1 (P) INHIBITOR
L3             3 DUP REM L2 (0 DUPLICATES REMOVED)
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d 13 1-3 bib abs

L3 ANSWER 1 OF 3 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1999416694 EMBASE
TI HMG-I(Y) recognizes base-unpairing regions of matrix attachment sequences and its increased expression is directly linked to metastatic breast cancer phenotype.
AU Liu W.-M.; Guerra-Vladusic F.K.; Kurakata S.; Lupu R.; Kohwi-Shigematsu T.
CS T. Kohwi-Shigematsu, Berkeley National Laboratory, University of California, 1 Cyclotron Road 70A-1118, Berkeley, CA 94720, United States
SO Cancer Research, (15 Nov 1999) 59/22 (5695-5703).
Refs: 88
ISSN: 0008-5472 CODEN: CNREA8
CY United States
DT Journal; Article
FS 016 Cancer
LA English
SL English
AB Base-unpairing regions (BURs) contain a specialized **DNA** context with an exceptionally high unwinding propensity, and are typically identified within various matrix attachment regions. A BUR affinity column was used to purify a doublet of M(r) 20,000 proteins from human breast carcinoma cells. These proteins were identified as the high-mobility group (HMG) protein, HMG-I, and its splicing variant, HMG-Y. We show that HMG-I(Y) specifically binds BURs. Mutating BURs so as to abrogate their unwinding property greatly reduced their binding affinity to **HMGI** (Y). Numerous studies have indicated that elevated HMG-I(Y) expression is correlated with more advanced cancers and with increased metastatic potential. We studied whether the expression of HMG-I(Y) responds to signaling through the heregulin (HRG)-erbB pathway and the extracellular matrix. HMG-I(Y) expression was increased in MCF-7 cells after stable transfection with an HRG expression construct that led cells to acquire estrogen independence and metastasizing ability. A high level of HMG-I(Y) expression was detected in metastatic MDA-MB-231 cells, but the expression was virtually diminished, and the metastasizing ability was lost after cells were stably transfected with an antisense HRG cDNA construct. HMG-I(Y) was also decreased in MDA-MB-231 cells when treated with a chemical **inhibitor** for matrix metalloproteinase-9 that led to a reduction of invasive capability in vitro. The level of HMG-I(Y) expression, therefore, is dynamically regulated in human breast cancer cells in response to varying types of signaling that affect metastatic ability, including the HRG-erbB pathway and those from the extracellular matrix.

L3 ANSWER 2 OF 3 LIFESCI COPYRIGHT 2000 CSA
AN 96:109121 LIFESCI
TI Induction of interferons and interferon-induced genes
AU Samuel, C.E.; Ozato, K.
CS Dep. Biol. Sci., and Grad. Prog. Biochem. and Mol. Biol., Univ. California, Santa Barbara, CA 93106, USA
SO BIOTHERAPY, (1996) vol. 8, no. 3-4, pp. 183-187.

ISSN: 0921-299X.
DT Journal
TC General Review
FS F; G
LA English
AB Type I IFNs (IFN- alpha , IFN- beta , and IFN- omega) are produced in most cell types upon virus infection and upon stimulation by dsRNA (with exceptions of embryonal carcinoma cells which are defective in expressing IFN genes). In addition, certain growth factors and lymphokines such as IL-1, colony stimulating factor (CSF), and TNF also induce type I IFNs. Both IFN- alpha and IFN- beta are capable of enhancing induction of type I IFN genes. Protein synthesis **inhibitors** such as cycloheximide often enhance levels of IFN mRNA, perhaps by inhibiting a labile repressor molecule. Type II IFN (IFN- gamma) is produced in T cells (TH1) and NK cells in response to stimulation with lymphokines such as IL-2 and IL-12. IFN- gamma is also induced by antigen stimulation through the T cell receptor/CD3 as well as by a costimulator CD28. Its production is down-regulated by IL-4. Induction of IFN genes involves primarily transcriptional activation. The promoter elements controlling virus activated transcription have been extensively studied for the IFN genes. Four domains (PRDI-PRDIV) that take part in activating the IFN gene expression have been identified within a 200 bp upstream region. In addition, negative regulatory elements that repress constitutive expression of the gene before virus stimulation map within this region. PRDI is similar to the ISRE present in many IFN inducible genes and is shown to bind IRF-1 and IRF-2 (interferon regulatory factors). Previous evidence indicates that IRF-1 plays an essential role in activating type I IFN genes. PRDI when placed on a heterologous promoter confers activation by dsRNA and by IFN. PRDII is an NF Kappa B site, which is activated not only after virus stimulation but also by various other stimuli that inactivate the **inhibitor** subunit, I Kappa B. This may involve activation of PKR. Cooperative activities of the closely juxtaposed elements in the IFN gene may be achieved by the amino acid motif of **HMGI**(Y) that apparently affects local **DNA** bending. Promoter regions in the IFN- alpha genes so far studied (IFN- alpha 1, IFN- alpha 2, IFN- alpha 4, IFN- alpha 11) reveal a conserved 42 bp purine-rich element which contains a GAAATG motif that resembles the ISRE. This element is important for induction by viruses. However, the role of IRF-1 and IRF-2 in regulating the IFN- alpha genes is still not clear: while transfection data indicate that IRF-1 upregulates IFN- alpha production. IRF-1 binds to this element with only a low affinity. The trophoblast IFN (IFN- tau) exhibits unique regulation. IFN- tau is expressed only in placenta, and does not respond to infection by viruses. Its promoter lacks a virus inducible element, but directs constitutive expression in placenta-derived cells.

L3 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2000 ACS
AN 1997:29520 CAPLUS
DN 126:55408
TI Molecular genetics of ergosterol biosynthesis in Ustilago maydis
AU Hargreaves, J.A.; Keon, J.P.R.; Croxen, R.
CS IACR - Long Ashton Research Station, Department of Agricultural Sciences, University of Bristol, Bristol, BS18 9AF, UK
SO Mod. Fungic. Antifungal Compd., Int. Symp., 11th (1996), Meeting Date 1995, 117-123. Editor(s): Lyr, Horst; Russell, Philip E.; Sisler, Hugh D.
Publisher: Intercept, Andover, UK.
CODEN: 63RYAG
DT Conference; General Review
LA English
AB A review with 22 refs. The application of fungal mol. genetics to study

sterol biosynthesis and ergosterol function in Ustilago maydis is considered, wi pecific ref. to genes encoding own target enzymes of sterol biosynt s **inhibitors** (SBIs). Using the ERG2 gene encoding a sterol isomerase, the ERG11 gene encoding 14.alpha.-sterol demethylase (14.alpha.-DM) and the **HMGI** gene encoding 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase as examples, the potential contribution of fungal recombinant **DNA** techniques to **inhibitor** mode of action studies and to fungicide development